Neural Modeling of Imitation Deficits

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This abstract addresses the question of human imitation through convergent evidence from neuroscience. We look at deficits in imitation following brain lesion, such as *apraxia*. We believe that looking at how imitation is impaired can unveil its underlying principles. We also take inspiration from numerous brain imaging studies to ground the functional architecture and information flow of our model. In the end we will use findings from monkey brain neurophysiological studies to implement the details of our processing modules.

We aim at developing a model of visuo-motor imitation using tools from neural networks and dynamical systems. The model should account for some of the behaviors observed in faulty imitation. At this stage we have implemented a somatotopically organized neural network with probabilistically impaired transfer of information that simulates lesions at the level of the parietal cortex (a brain center for sensorimotor integration). To validate the model against human motion experimental data, we conduct, in collaboration with the Geneva University Hospital (HUG), kinematic studies with brain damaged adults specifically disabled in gesture imitation. The model will motivate the realization of computer-based rehabilitation tools.

Introduction. Apraxia is generally defined as the inability to perform voluntary movements that cannot be explained by elementary motor, sensory or cognitive deficits (not caused by weakness, ataxia, akinesia, deafferentation, inattention to commands or poor comprehension). Some apraxic patients are impaired for imitation of meaningless gestures, which is believed to test the integrity of a direct route from visual perception to motor control, not mediated by semantic representations or verbal concepts. Knowledge about the human body is also relevant as apraxic patients are unable to map body configurations to their own body or to a mannikin[2]. Kinematic studies show that patients exhibit either a completely normal kinematic profile, but abnormal final position; or kinematic abnormalities (slowing and repeated changes of direction of movement), with correct target [4]. Spatial parapraxias seem to arise from a basic deficit that might concern the mental representation of the target position and kinematic abnormalities from the strategy of online visually controlled movements that cope with it.

Experimental Study. A seminal study of imitation of meaningless gestures [3], by Goldenberg, was of particular interest to us (Fig. 1). A patient that suffers from a disconnection between the two hemispheres (following callosal lesions) was asked to imitate hand postures in relation with the head. The study shows that the pattern of errors varies as a function of the visual field to which the stimuli to imitate are displayed and the hand used to execute the imitative movement. Imitation was perfect only in the right visual field - right hand condition, indicating a lateralization of the processing to the left hemisphere and a non-uniform information flow across the two hemispheres.

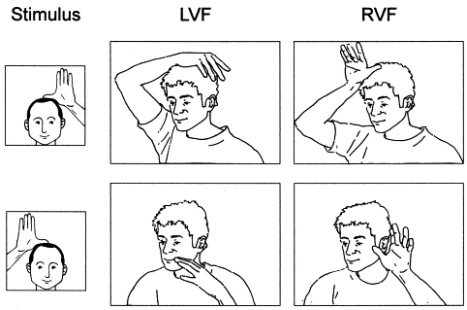

Figure 1: Goldenberg's experiment of imitation of meaningless gestures and an example of errors made by the patient, from [3].

As we did not have access to data on errors in imitation of apraxic patients (lesion studies provide only statistical data of the correctness of the imitation), we decided to replicate Goldenberg's experiment of imitation of meaningless gestures in collaboration with the HUG [3]. As we were interested in providing a quantitative data of the deficit, we extended the experiment to record the movement kinematics and hand posture with motion tracking sensors.

Computational Model. A neural model of *imitation of meaningless gestures* would encompass several regions dedicated to specific functions, shown in Fig. 2. We concentrate our modeling work on the parietal cortex, considered to be the center for visuo-motor

and multimodal (somatosensory, visual, auditive and vestibular) integration (lower-order computations than in the frontal cortex). Moreover, lesions in the parietal cortex lead to imitation deficits. We have implemented

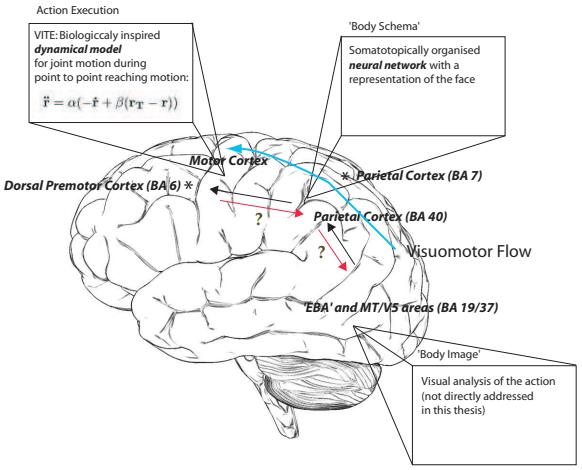

Figure 2: Our neurocomputational model of imitation of meaningless gestures. The functional architecture and connectivity of our model is inspired by data from human brain imaging studies [1, 5]. We have modeled area BA 40 (a sensorimotor 'body schema' module) as a face somatotopically organized neural network, locus of simulated lesions. An 'action execution' module (in the motor cortex) is necessary for validating the model against experimental data and implements a biologically inspired dynamical model for reaching (VITE).

a computational model of this region to simulate *focal and diffuse* lesions of the transfer of information between its left and right parts (see Fig. 4). We decided to use leaky integrate and fire neurons, which is a simple dynamic model that accounts for variations in the neuron membrane potential m_i of neuron N_i over time:

$$\tau_i \cdot dm_i/dt = -m_i + \sum w_{i,j} x_j \qquad (1)$$

where x_j represents the neuron's short-term average firing frequency, τ_i is a time constant associated with the passive properties of the neuron's membrane, and $w_{i,j}$ is the synaptic weight of a connection from neuron N_j to neuron N_i. The input is both visual (the stimulus to imitate) and somatosensory (a departing posture and target in terms of relations of body-parts). Therefore it seemed natural to have a somatotopic organization of the information, as is the case in several parietal regions. We trained a neural network to respond to particular regions of the face (Fig. 3), using Kohonen's algorithm. In the end we obtain a somatotopic representation of the face: parts close to each other on the face have close representations in the neural space and parts more important than others (such as the eyes and the mouth) have larger representations. As the patient converges to the correct response with time, we

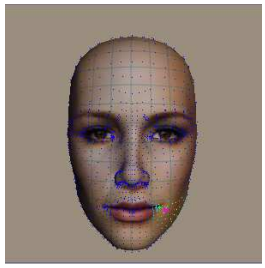

Figure 3: A model of the face with tactile sensors, which are the input to our SOM.

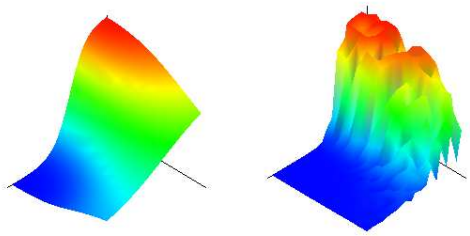

Figure 4: Neural network applet for training the somatotopically organized network (SOM) related to the face. The activation from neurons in the left network is transmitted to the right network with probability p_i from Eq. 2 simulating the brain lesion.

decided to simulate the lesion in a probabilistic way. We suppose that the information does not always fail to transfer, thus each neuron is assigned a probability p_i of firing and transmitting the value of the membrane potential to the corresponding neuron in the other hemisphere (related to the severity of the lesion):

$$P(x_j = (1 + e^{-m_j + b_j})^{-1}) = p_i, \quad x_j = 0 \text{ otherwise} \qquad (2)$$

with $0 \leq p_i \leq 1$. Varying lesion parameters (type, size, locus and severity) induce different patterns of errors.

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